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# Takotsubo cardiomyopathy following a simple partial seizure

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## ABSTRACT

A 67-year-old woman presented to the emergency department for a simple partial seizure of her left upper and lower limbs that lasted for 1 hour and ultimately resolved before her presentation. She had no history of coronary artery disease, and her neurological exam was normal. Five hours later, she complained of chest pain. An electrocardiogram showed ST segment elevation in the lateral leads, and her troponin level was increased. She was diagnosed with takotsubo cardiomyopathy. This case reflects the brain-heart connection and is the first reported case of takotsubo cardiomyopathy following a simple partial seizure.

**KEYWORDS** Echocardiography; epilepsy; neurogenic stunned myocardium; takotsubo cardiomyopathy; ultrasound

Stress-induced cardiomyopathy (SIC) (takotsubo cardiomyopathy or broken heart syndrome) is a condition that occurs following physical or emotional stress. It can mimic acute coronary syndromes because of its similar symptoms and electrocardiogram (ECG) features. Seizures are a well-known trigger of SIC. Seizures are classified as focal and generalized. Focal seizures can be further stratified into simple or complex, depending on the presence or absence of consciousness, respectively. Complex focal and generalized seizures are the two main types that can cause SIC. We present a rare case of SIC following a simple partial seizure in a patient with familial meningiomatosis.

## CASE REPORT

A 67-year-old woman presented to the emergency department for abnormal movements of her left upper and lower limbs that lasted for 1 hour and resolved ultimately before her presentation. The patient and her accompanying relative reported no confusion, loss of consciousness, or head trauma. Her history included familial meningiomatosis diagnosed 3 years earlier and treated with partial surgical resection. Her medications included sodium valproate. She had no history of coronary artery disease. On presentation, the neurological exam was normal. A brain computed tomography scan showed an increase in the size of the meningiomas with a right anterior parafalcine edema, without intracranial hemorrhage. We established the diagnosis of epilepsia partialis continua, and she received diazepam with a loading dose of sodium valproate.

Five hours later, she complained of an oppressive chest pain irradiating to the interscapular area. Vital signs were normal. An ECG showed sinus rhythm with ST elevation in the lateral leads (DI and aVL) with a mirror image in the inferior leads (DII, DIII, aVF) *(Figure 1)*. Laboratory tests revealed a highly elevated troponin T level (1372 ng/L), and an echocardiogram showed apical ballooning with akinesis, hyperkinetic basal segments, and a reduced ejection fraction *(Figure 2a, Video 1)*. Coronary angiography showed no significant abnormalities *(Video 2)*. We diagnosed SIC and put her on a beta-blocker. A follow-up echocardiogram 72 hours later showed a significant decrease in the apical ballooning and improved systolic function *(Figure 2b, Video 3)*. The troponin T level decreased to 1078 ng/L 24 hours after her presentation and 849 ng/L 72 hours later.

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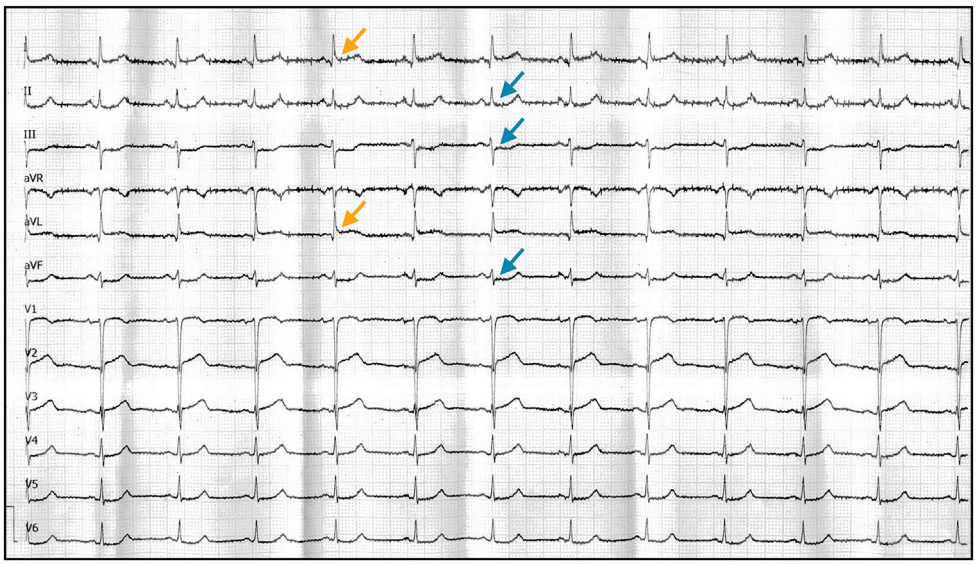


**Figure 1.** Electrocardiogram on presentation showing ST segment elevation in the lateral (orange arrows) and septal leads with mirror image in the inferior leads (blue arrows).

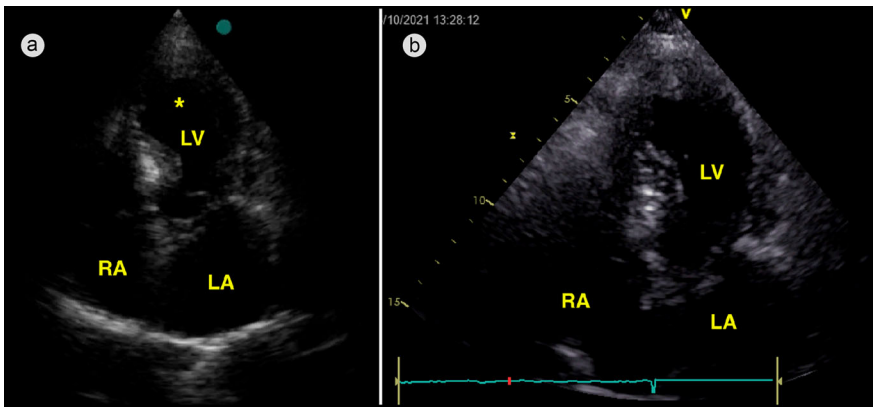


**Figure 2.** Echocardiography, apical four-chamber view **(a)** on presentation, showing apical ballooning (yellow asterisk) and **(b)** 72 hours after presentation, showing a significant decrease in the apical ballooning. LA indicates left atrium; LV, left ventricle; RA, right atrium.

**Table 1. Main characteristics and differences between takotsubo cardiomyopathy and neurogenic stunned myocardium**

| Variable | Takotsubo cardiomyopathy | Neurogenic stunned myocardium |
|---|---|---|
| Cause | Emotional or physical stressors | Acute brain injury including SAH |
| Sex predominance | Female | Female |
| Clinical presentation | Chest pain | Heart failure |
| ECG features | ST elevation; prolonged QT; T-wave inversion | Prolonged QT; T-wave inversion |
| Echographic features | Apical ballooning due to mid-ventricular and apical dyskinesia and hyperkinetic basal segments | Apical sparing RWMA; basal and mid-ventricular hypo/akinesia |
| Troponin T peak | 1.05 | 0.4 |
| Mechanism | Systemic release of epinephrine | Local release of norepinephrine |
| Receptors | Activation of beta-2 receptors in apical segments | Activation of beta-1 adrenergic receptors and sympathetic nerve terminals at the base |

ECG indicates electrocardiography; RWMA, regional wall motion abnormalities; SAH, subarachnoid hemorrhage.

## DISCUSSION

In acute neurologic conditions, the brain-heart connection can include a spectrum of manifestations that comprises cardioinhibitory, vasodepressing, and myocardial syndromes. SIC and neurogenic stunned myocardium (NSM) are two myocardial syndromes reported after neurological events. The clinical, echocardiographic, and electrocardiographic features help us differentiate between these two entities *(Table 1)*.[1] It is believed that the pathophysiology of SIC involves the systemic release of epinephrine and activation of beta-2 receptors in apical segments while, on the contrary, there is a local release of norepinephrine in NSM and activation of beta-1 receptors and sympathetic nerve terminals at the base. However, others consider NSM and SIC as having the same pathophysiologic phenotype.[2]

Stöllberger et al screened the literature for all SIC cases occurring in the setting of seizure. In 75% of cases, there was an underlying neurologic disease or a direct cause of epilepsy, and generalized tonic-clonic and complex partial were the most commonly involved seizure types, with no report of SIC following a simple partial seizure.[3] This unique case reports the first SIC following a simple partial seizure in a patient with a prior history of partially resected meningiomatosis. The long seizure duration in our patient, which defines epilepsia partialis continua, may have caused SIC.

## SUPPLEMENTAL MATERIAL

**Video 1.** Transthoracic echocardiography apical four-chamber view on presentation showing apical ballooning with akinesis and hyperkinetic basal segments.

**Video 2.** Coronary angiography showing no significant abnormalities.

**Video 3.** Transthoracic echocardiography apical four-chamber view 72 hours after presentation showing a significant decrease in the apical ballooning.

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